ASCARIDES IN THE BILE-DUCTS SIMULATING GALLSTONE SEIZURES.

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Intestinal worms are not considered common causes of biliary colic; they are, perhaps, not sufficiently considered in this role, for when we remember that the natural habitat of these parasites is the small intestine, and that not infrequently do they pass into the common duct and there effectually occlude the lumen of that canal and completely obstruct the flow of bile, it is obvious that their presence would give rise, quite naturally, to the complex chain of symptoms noted in the occlusion of that duct from other causes.

Where the natural current of normal bile is not interfered with it is a sterile fluid. This has been proven experimentally on dogs by Netter[1] and confirmed by Gilbert and Girade,[2] and further confirmed by Naunyn,[3] who reports a case wherein the bile was sterile two hours after death.

Bile has also been regarded an antiseptic, and to this property has been ascribed the function of preventing decomposition during digestion; but this premise has failed to receive sufficient proof, while on the other hand it has been proven that it does not possess any antiseptic properties. In the report of a case of biliary fistula Mayo-Robson observed that the absence of bile from the intestine for a period of fifteen months did not lead to any irregular fermentation processes, and concludes "that the alleged antiseptic effect of bile on the feces is probably imaginary."[4] Professor Birch has reached the same conclusion[5] after numerous cultivation experiments.

So much, then, in consideration of normal bile when its natural current is not impeded.

In emphatic contrast to the researches noted above we know, from equally able investigators, that when the flow of bile along the ducts is for any reason arrested, micro-organisms *usually do* invade the gall-bladder, on the hypothesis that they gain entrance either from the blood or from the intestines.

Charcot and Gombault[6] were the first to demonstrate this fact by ligating the common duct in dogs; later this was confirmed by Netter,[7] who, after an aseptic ligature of the common duct in dogs, found that both staphylococcus and bacillus coli commune could be cultivated from the stagnant bile. Bouchard[8] cultivated the bacillus coli commune from the bile of cases suffering with disease of the biliary tract which was of sufficient severity to produce inflammatory changes on the mucous lining of the ducts, thus obliterating the lumen of the canal, and in consequence producing a stagnation of the bile.

In answer to the query, "How do these bacteria reach the gall-bladder?" Mayo-Robson[9] gives three possible sources, viz.: (*a*) through the bile-ducts, or (*b*) from the blood, or (*c*) directly through the wall of the gall-bladder.

It is, then, only in diseased conditions that these micro-organisms are present in bile, and it seems that the biliary apparatus will tolerate a moderate degree of inflammatory involvement and be perfectly able to maintain its integrity, providing the flow of bile is not interfered with, but cannot withstand the closure of the chief duct and resist the invasion of the bacteria.

This fact, then, becomes the pivotal point which must determine the causes of gall-bladder infections. It makes no difference whether the bile current is stagnated by an inflammatory process involving the duct to the degree of occluding its patency, or whether there is an impacted gallstone in the common duct, or whether an ascaris has wandered from its habitat and entered the common duct, or the simple aseptic ligature, experimentally performed; the result is the same, the flow of normal bile is prohibited, and bacterial invasion is certain to be a consequence.

It is not surprising, then, that the case cited below was admitted to the hospital and the opinion given of probable impacted gallstone in the common duct:

A. S., aged thirty-four years, Austro-Hungarian. Engaged in railroad grade work. Lives in a temporary camp with other men of his class. Surroundings very unhygienic. Food improperly prepared and of poor quality.

April 12, 1904. *Present History.* Complains of pain in stomach, no appetite, tongue coated and foul, fetid breath. Has had attacks of severe colic, lasting a few hours, leaving a sense of soreness in epigastrium. Temperature, 99° F.; pulse, 80; anxious facial expression.

19*th*. Admitted to Mercy Hospital. Deeply jaundiced; mucous membranes of the mouth and conjunctiva yellow; sclerotic membranes of both eyes deeply jaundiced. Hepatic area of dulness much increased, border of liver extending 6 cm. below costal arch; exceedingly tender on slight pressure over site of gall-bladder, tumor evident between the costal margin of ninth rib and umbilicus. Pulse, 60° F.; temperature, 98.4; respiration 16. Frontal headache, anorexia, and eructations of gas. Constipated. Urine: dark-brown colored; specific gravity, 1028; reaction, acid; chlorides, 16 per cent.; phosphates, 8 per cent.; sulphates, 8 per cent.; albumin, none; sugar, none. Biliary pigments distinct by Ulzmann's test. Biliary acids were detected by Dr. Oliver's test,[10] and estimated by his standard table as follows: To 4 c.c. of Oliver's solution 1.25 c.c. of urine was added; a very distinct milkiness appeared at once.

In the second trial 4 c.c. of the test solution was added to 1 c.c. of urine, with prompt milkiness. In the third trial 4 c.c. of the test solution was added to 0.60 c.c. of urine, resulting in no immediate reaction, but in a short time slight milkiness occurred. According to Oliver's table of standards this would indicate a percentage increase of 600. Stool almost white, and about the consistency of putty.

Patient was ordered a liquid diet, and, as was suggested by Mayo-Robson,[11] he was given calcii chloridi, 2 grams three times daily, to lessen the tendency to hemorrhage during the operation. Calomel 0.65 every hour for three doses, follow at once with $MgSO_4$, 15 grams in half-glass of hot water.

20th. Pulse, 49; temperature, 97° F.; respirations, 16. No bowel movements. Urinated about 300 c.c. during the night, highly colored. Pain quite severe in stomach. Deep jaundice; headache. Calomel 0.65 every hour for three doses, follow at once with $MgSO_4$, 15 grams in hot water. Calcii chloridi, 2 grams three times daily.

21st. Pulse, 58; temperature, 97.8° F.; respirations, 16. Bowels have not moved; urinated four times. Complains of headache. Deeply jaundiced. Pain constant. Calomel reordered as above, followed by $MgSO_4$. Calcii chloridi continued. Prepared for operation.

23d. Pulse, 52; temperature, 98.2° F.; respirations, 16. No breakfast. Had four bowel movements, clay colored, thin and offensive; the second movement occurred at 5 A.M., and was allowed to remain in the bed-pan and placed in the closet. The nurse reported a long, brown body in the evacuation, and Dr. J. M. Crowley, the assistant superintendent of the hospital, examined the evacuation and secured a live ascaris lumbricoides 28 cm. long. On my arrival at the hospital, Dr. Crowley and I examined the evacuation for the ovæ, but none were found. It was thought that possibly this worm was responsible for the symptoms manifested, and operation was postponed. Santonini, 0.65, with calomel, 1.95, was ordered at noon, to be repeated at 9 P.M., and followed by Ol. ricini 30 c.c. in the morning.

24th. Slept well. Bowels moved four times freely, clay colored, thin; urine dark. Deeply jaundiced.

The subsequent history is not of especial interest. In a few days the pain subsided, the liver area of dulness became normal, no pain on pressure could be elicited, tongue cleaned, appetite returned, jaundice gradually faded, and he was discharged May 5, 1904.

It might be of interest to note that, while the patient began to improve in every way and the appetite was fully restored, while the bowels moved regularly every day, it was not until the day previous to his discharge from the hospital that the feces resumed their natural color, and when the coloring of the bile did appear in the stools it was not gradual but spontaneous; the evacuation preceding the one of natural color was as cream colored as they were when he first entered the hospital.

Mertens[12] has reported forty-eight cases of ascarides in the bile-ducts, but in only five cases were the symptoms such as would suggest the presence of gallstones, and in but two of these was the cause determined during life.

In his own case, a woman of thirty years of age was admitted to a hospital following the third attack of biliary colic, which began with pain in the gall-bladder region, associated with vomiting, and followed by deep jaundice; both liver and spleen were increased in size. The patient rapidly became emaciated for seven weeks, at the end of which time she passed two partially macerated round worms, and she rapidly improved and ultimately recovered.

When we consider that these worms inhabit the upper portion of the small intestine, and recall the migratory nature of the parasite, the wonder is not that they occasionally wander into the common duct, but that they are not found there more frequently than observers would lead us to believe.

Their presence in the bile-duct certainly interferes with the normal flow of the bile and invites bacterial invasion by stagnation of that fluid, and I believe that in the cases cited above such an infection occurred, and this premise is substantiated by the tardy appearance of the bile in the stools. There is no doubt but that the gall-bladder and the bile-ducts were infected and suffered inflammatory hypertrophy sufficient to completely obliterate the lumen of the common duct, and even after the removal of the provoking cause were slow to repair this damage and functionate normally. The pain symptom further emphasizes the truth of this statement.

Kehr[13] states that pain in gallstone disease is not due, as is commonly supposed, to the pressure of a hard substance held in a soft mucous-membrane tract, either in the cyst or the ducts; the mere presence of stone in these passages may interfere with the normal currents of the bile, and that bacterial invasion which follows this impeded current invade the gall-bladder and its ducts. The result of this inflammation is the softening of the mucous membrane of the gall-bladder and the ducts, and the transudation of serous or purulent fluid into these hollow organs; at first the transudation takes place by reason of the increased circulation attracted to this site by the bacterial invasion of the coats of either the gall-bladder or the ducts, and when first poured out is a serous fluid; later, as the bacterial invasion increases, this serous fluid is readily converted into a purulent fluid.

In either case the gall-bladder and its appendages are distended, and the nerve endings throughout its surface are put upon the stretch and dragged upon, and this gives the symptoms of pain, and is solely excited by the distention of the walls of the gall-bladder and ducts.

It is not the character of the fluid in those hollow organs that determines the pain, but the fact that *any* fluid has produced distention sufficient to violate the nerve endings.

Kehr[14] mentions a simple interesting experiment in proof of this statement: following an operation on the gall-bladder where there is a fistula established, inject into the gall-bladder physiological salt solution; the moment distention occurs, complaint will be made by the patient of severe cramp in the stomach.

The jaundice of the case was very pronounced; all visible tissues were deeply stained; the skin was dry and harsh. This color appeared quite abruptly, following shortly after my first examination on April 12th, and was of the same deep color that was so characteristic a feature until the passage of the worm on April 23d; after this the color gradually faded.

It seems that these round worms are very commonly found in autopsies upon laborers of this class, especially among the Italian. Dr. F. F. Agnew, formerly of Blockley Hospital, Philadelphia, stated to me in a conversation recently that in a number of autopsies during his service he cannot recall an instance when these worms were not found in this class of laborers. Thus it would seem that the mode of living and the sanitary surroundings had a determining influence.

The hospital record shows that this patient complained bitterly of pain, and was not able to sleep well from the date of his admission, April 19th, until the date on which he passed the worm, April 23d. After that date his appetite improved and he slept well, and when seen he would make deep pressure over the former site of his pain, which was a little to the right of the epigastric median line, and say in English, "No pain," and shake his head vigorously to emphasize the statement.

There were no special symptoms that might lead one to suspect the presence of ascarides; the clinical picture was purely one of biliary colic, and, combined with the quickly developing jaundice and the continued pain, presented a typical case of obstruction of bile from whatever cause.

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